

THE AUTHORS REPLY

Dr. Morgan (1) states that our investigation was not initiated with an explicit hypothesis. Quite the contrary. This research investigation (2) within the Multiple Risk Factor Intervention Trial (MRFIT) was carefully planned and undertaken because of the growing body of evidence that environmental tobacco smoke is a health hazard to nonsmokers. Reports that document exposure of nonsmokers to environmental tobacco smoke, such as elevated carboxyhemoglobin or cotinine in exposed persons, as well as reports of a possible relation between environmental tobacco smoke and diseases such as lung cancer, pulmonary disease, and coronary heart disease prompted this investigation. An advantage of large scale clinical trials is that data are often collected which can be used to investigate other research questions. Our research hypothesis was formulated to utilize data that were collected in the MRFIT for another purpose. The MRFIT group had collected data on smoking habits of wives for all of the 12,866 participants prior to this study of the relation between environmental tobacco smoke and disease. These data were collected not because of an interest in environmental tobacco smoke, but rather because we believed the wife's smoking behavior might impact the participant's ability to change risk factors; in particular, the ability to quit smoking for participants who were smokers.

The endpoints presented in our paper—coronary heart disease (CHD) death, fatal or nonfatal CHD event, and death from any cause—were the endpoints used for the primary MRFIT trial. Dr. Morgan is correct in observing that the CHD deaths are counted when considering the endpoints fatal or nonfatal CHD event and death from any cause. The intent was not to repeatedly test the difference between the same proportions, but to investigate if the smoking behavior of the participant's wife was related to these major MRFIT endpoints defined at the beginning of the study.

The focus of our paper (2) was on MRFIT men who had never smoked tobacco products. We repeated the table of relative risk estimates for all nonsmokers (which included never smokers and ex-smokers who quit prior to entry into the MRFIT) to provide data for comparisons with other studies which may not have such detailed lifetime smoking histories. The lower p value for the endpoint "death from any cause" in table 9 primarily reflects increased sample size and not strength of association. The hypothesis that the relative risk for this endpoint would be higher if the ex-smokers were considered alone is false. The relative risk is 1.60 ($p = 0.08$, 95 per cent confidence interval = 0.95-2.69), compared with 1.96 for never smokers (table 8) and 1.72 for all nonsmokers (table 9).

Dr. Katzenstein (3) suggests lack of homogeneity between the men who had never smoked tobacco products whose wives smoked versus those whose wives did not smoke. As noted in our paper (2) one of the strengths of the MRFIT data set was the large amount of information available regarding the biologic, social, and behavioral characteristics of the participants at entry to the trial. Baseline characteristics of men whose wives smoked and men whose wives did not smoke were similar, as we noted in table 2 of our paper

and as observed by Martin et al. (4). The significant differences were men whose wives smoked weighed 4.2 lbs (1.9 kg) more, consumed 2.1 more drinks per week, and had 0.5 years less formal education, than men whose wives did not smoke. Weight was not associated with coronary heart disease death or total mortality in the MRFIT study (5). Adjustment for baseline differences in weight, alcohol consumption, and education (used as a measure of socioeconomic status), as well as age, blood pressure, and cholesterol did not change the relative risk estimates appreciably.

Clearly, however, not every variable that might possibly differ between the husbands of women who smoke and those who do not smoke were measured. There are almost certainly social and behavioral differences between a man who is a lifetime nonsmoker married to a woman who smokes, and a man married to a woman who also does not smoke. It is possible that a man who does not smoke married to a wife who smokes makes behavioral changes because of the habit of his wife which increases his risks of death, independent of the known toxic chemicals in the environment from his wife's cigarette smoke. The ideal study, randomizing nonsmoking men to smoking or nonsmoking wives, cannot be done.

We agree with Dr. Katzenstein that the lack of a dose-response relation makes the pulmonary function data weaker. The difference in FEV₁ between men whose wives smoke 1-19 cigarettes per day and those whose wives smoke 20 or more cigarettes per day is not significant so the dose-response relation is lacking, not reversed. In view of our carbon monoxide and mortality findings, along with other studies referenced in our paper, we see no reason to alter our conclusions.

REFERENCES

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3. Katzenstein AW. Re: "Effects of passive smoking in the Multiple Risk Factor Intervention Trial." (Letter.) *Am J Epidemiol* 1989;129:227.
4. Martin MJ, Svendsen KH, Kuller LH. Nonsmoking men married to smokers are similar to nonsmoking men married to nonsmokers. (Abstract.) Society of Behavioral Medicine, 7th Annual Scientific Sessions, San Francisco, CA, March 5-8, 1986.
5. Multiple Risk Factor Intervention Trial Research Group. Relationship between baseline risk factors and coronary heart disease and total mortality in the Multiple Risk Factor Intervention Trial. *Prev Med* 1986;15:254-73.

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